INHIBITION AND DISINHIBITION IN HYPNOSIS

PETER L. N. NAISH

The Sackler Centre for Consciousness Science, University of Sussex

ABSTRACT

This small-scale, quickly-administered study was designed as an initial exploration of the extent to which general inhibition plays a part in hypnosis. Some models of hypnosis emphasize the strategic use of prefrontal disinhibition, while others suggest that frontal regions become less involved overall, so that disinhibition is general rather than focused. A stop signal reaction time task was used to assess the level of inhibition available in high and low susceptible participants, both waking and hypnotized. Results implied that hypnosis increases inhibition for the highly susceptible. From this it is concluded that any disinhibition must be localized and strategic.

Keywords: consciousness, altered states, hypnotic susceptibility, inhibition, stop signal reaction time

INTRODUCTION

The advent of brain scanning has been of assistance in showing that hypnosis is more than just a simple blend of compliance to suggestions and 'imagining along' with them (e.g. Naish, 2013). As a result, although there may still be some dissenting voices (see Kirsch, et al., 2011) it has become reasonable to consider hypnosis to be an altered state of consciousness. However, the various forms of brain scanning and monitoring available to us are, as yet, unable to provide full explanations of consciousness itself, so to describe hypnosis as some kind of modification to consciousness is not entirely helpful.

What we do know, of both hypnosis and consciousness more generally, is that the prefrontal cortex (PFC) plays a key role. The work of van Gaal and Lamme (2012) has helped to draw together and explain several significant observations. First, it is known that a briefly displayed word, shown too quickly for us consciously to know what we saw, can nevertheless influence our behaviour; clearly, analysis has taken place outside conscious awareness. Brain scanning in this situation shows that activity spreads forward through the brain, starting at the back at the onset of the stimulus, in the visual cortex, then moving through the processing sequence, getting closer to the front of the brain. Crucially, when the stimulus is very brief, the activity never reaches as far forward as the PFC. Interestingly, a slightly longer-lasting stimulus that does result in PFC activity still does not necessarily result in conscious awareness. That seems to occur only after the PFC initiates backward-acting signals. These appear to result in the phase-locking of activity pertinent to the experience (Doesburg et al., 2008).

Phase-locking is analogous to having different sections of an orchestra (but not all of it) following the conductor. Because the brain is highly modular the various aspects of an experience

will be represented by activity in different regions; for example the round shape of an orange, its colour and its smell will each be recognized in different brain areas. Nevertheless, the conscious experience is of a unified whole and scanning reveals that oscillations in those different regions remain locked in step. The synchronization is achieved by long-range feedback via neural tracts originating in the PFC; without this there seems to be no conscious awareness.

Imagining and remembering appear to utilize the same feedback pathways from the PFC. In this case, rather than controlling activity caused by external stimuli, the PFC is actually generating activity. One theory of why humans developed consciousness is based upon this ability to re-activate regions of brain that were no longer receiving external information. Baumeister & Masicampo (2010) proposed that this was very valuable to a social animal, since it would permit the repeated 're-running' of a social interaction, so enabling the ruminator to fine-tune future encounters. Scanning someone who is remembering or visualizing reveals activity that is very similar to that observed when the stimuli are actually present. Significantly, the similarity is much enhanced when visualization occurs in hypnosis (Szechtmanet et al., 1998; Kosslyn, et al., 2000). Comparison with the experiences of schizophrenia can help to explain why hypnosis may enhance the sense of reality.

Schizophrenia patients exhibit lower than normal levels of phase locking (Haig et al., 2000) and this appears to be the result of poor connectivity with the PFC (Lawrie et al., 2002). Perhaps an even more significant deficit is that, along with the excitatory links used for phase-locking or visualizing, the patients lose inhibitory connections too (Shergill et al., 2005). These are used to reduce activity in areas of the brain that are not contributing to conscious awareness (Buehlmann & Deco, 2008) especially those resulting from the person's own actions. Thus, when a healthy person moves their arm, although the receptors in the joints will be stimulated, little corresponding activity is seen in the brain. This low response is due to the arm movements being self-initiated, so making it possible to predict the potential neural responses and inhibit them. In contrast, if a person has their arm moved by someone else, then prediction is not possible and a clear, uninhibited signal is observed in the brain. Schizophrenia patients fail to inhibit self-generated signals so, presumably as a consequence, have the feeling that their actions are being controlled by external forces. Brain scanning hypnotic 'highs' reveals that, as with the patients, they exhibit reduced phase-locking (Fingelkurts et al., 2007). Moreover, when experiencing things 'happening by themselves', such as in the arm levitation test, they fail to inhibit the signals, just as if outside forces were moving the arm (Blakemoreet al. 2003).

The foregoing is a clear indication of the importance of the PFC in the generation of both normal and atypical conscious experiences. Although a more detailed understanding of the processes involved would permit a more precise description of hypnotic mechanisms, nevertheless it seems reasonable to claim that changes in the excitatory and inhibitory behaviour of the PFC underpin the experiences of hypnosis. Since people exhibit a range of hypnotic abilities it may be supposed that the extent of the changes in PFC activity varies between individuals. Plausibly, the type of change could differ too, with some people perhaps very good at reducing inhibition, so making actions feel involuntary, while others may be better at manipulating excitatory signals in order to generate hallucinations. These conjectures imply considerable mental versatility, with PFC activity being finely tuned to achieve the desired outcome. As Spanos frequently emphasized (e.g. Spanos, 1986) people in hypnosis do what they believe people in hypnosis are meant to do. Thus, the PFC control is strategic and must involve a good deal of the executive, planning function associated with that region.

In contrast to the above, a number of researchers have associated hypnotic effects with a reduction in executive function, characterizing this as a state of hypofrontality. Semmens-Wheeler, Dienes and Duka (2013) present a good summary of the evidence suggesting a reduction of PFC involvement, and contribute their own finding that alcohol increases hypnotic susceptibility. Participants in their study, who had consumed a quantity of alcohol equivalent to approximately 500 ml of wine, rated their hypnotic experiences as more intense than those who had consumed placebo. Other tests confirmed that the alcohol had, as expected, impaired frontal lobe activity, so the authors concluded that hypnosis is associated with a reduction in frontal involvement. However, it is difficult to generalize from this finding, for there are a number of caveats, most of which the authors acknowledge. Importantly, it is not entirely clear what exactly is being facilitated or disrupted by the alcohol. It is obvious that increasing levels of intoxication would eventually impair hypnotic responding, so there is presumably some optimum level which permits the best hypnotic response. Whether that level impairs excitatory and inhibitory processes equally is unclear. It is generally assumed that alcohol disinhibits, and one of the tests used by Semmens-Wheeler et al. (2013) did confirm that participants in the alcohol condition had a reduced ability to inhibit. On the basis of the account given earlier, this might be expected to permit inappropriate activity in the brain, leading to the erroneous experience that the hypnotic effects were happening without volition. However, a generalized reduction in inhibition would tend to raise the overall activity across the brain; that is not what brain scanning in hypnosis shows, at least not for hypnotic 'highs'. McGeown et al. (2009) showed that the so-called default mode activity (the background activity when there is no specific task to be performed) was reduced in 'highs' when hypnotized. If these participants sometimes used disinhibition, as the results of Blakemore et al. (2003) would predict, then they must have been able to deploy the strategy very selectively. This is not what would be expected when alcohol was used as the disinhibiting agent. It should be noted that the McGeown et al. participants were 'highs', whereas those in the Semmens-Wheeler et al. study were deliberately selected to be only moderately susceptible, to allow for either enhanced or impaired susceptibility to be registered. It is not known whether people who are moderately susceptible simply do less of whatever 'highs' do, or whether the latter have a different way of achieving their responsiveness. The experiment to be reported here attempted to determine what 'highs' were doing – specifically looking for evidence of general disinhibition.

The test used was similar to the one employed by Semmens-Wheeler et al. (2013) when checking that alcohol had reduced the ability to inhibit behaviour; it is called the Stop Signal Reaction Time (SSRT) task. A participant has to respond as quickly as possible to a signal, but has to withhold the response if a second signal indicates *Stop!* That stop signal is delivered a little later than the first trigger signal, and if it is too much later the participant is unable to prevent him- or herself from responding. Those who have a reduced ability to inhibit behaviour, for example through intoxication or having a condition such as obsessive compulsive disorder, require the stop signal to be presented with relatively little delay. In this experiment participants were to be tested in and out of hypnosis, to determine whether their levels of inhibition changed between the two states.

METHOD

PARTICIPANTS

Fourteen students attending an Open University Summer School volunteered to take part, having been selected following testing with the Harvard Group Scale of Hypnotic Susceptibility (Shor & Orne, 1962). There were seven 'highs' and seven 'lows', each group comprising six female and one male participant. All were fully informed about the nature of the study, although not its precise purpose; they were merely told that it was 'looking to see in what ways the brain performed differently in hypnosis'. Since the testing diverted students from their primary objective at Summer School the test procedures were designed to be as brief as possible.

STIMULUS PRESENTATION

Stimuli comprised large block arrows, pointing either left or right, presented on a laptop computer. Participants responded by pressing a left- or right-hand key on the computer keyboard. Presentation began with only the shaft of the arrow appearing in the centre of the screen, comprising a broad, black, horizontal line. After a random delay of between 1 and 2 seconds a black arrow head was added to one end or the other, with equal probability. At this point reaction timing began. Participants were instructed to respond to the direction as quickly as possible, but on 30% of presentations the black arrow turned red. This was the stop signal, and if it occurred participants were not to make a response.

Initially, the delay between arrowhead presentation and the stop signal was 200 ms, but this delay tracked the success or otherwise of the participant in withholding the response. After two consecutive successes the delay was lengthened by 64 ms, but after two consecutive inhibition failures the change was reversed and the delay reduced. At each reversal the step size was halved: 32, 16 ms and so on. In this way the delay was adjusted towards a value at which the participant had a 50% chance of correctly withholding the response.

If no response was made the screen was cleared after 3 s from stimulus onset; alternatively, if there had been a response the screen went blank 750 ms after the key was pressed. After a further 750 ms the sequence was repeated, until a total of 72 stimuli had been presented. At the end, the grand mean of all reaction times (RTs) to black arrows was calculated. The stop signal delay (SSD) was taken to be the mean of the most recent delays resulting in a) success and b) failure. Thus, if a participant successfully withheld a response with a delay of 320 ms, but responded when the delay was 336 ms, then the SSD was assumed to be 328 ms. The SSRT was calculated as mean RT – SSD. A larger value of SSRT implies less inhibition.

EXPERIMENTAL PROCEDURE

All participants were first given 24 practice stimuli, to familiarize them with the task. Following this, four from each group went on to be tested in the waking condition first, while three were hypnotized before continuing. The induction consisted of progressive relaxation and guided visual imagery, following which participants were instructed that it would be easy for them to open their eyes and perform the task they had seen before. After testing they were told to close their eyes, then were given formal waking instructions. After the first sequence, participants repeated the procedures in the other condition.

Table 1 Mean and (SD) SSRT values (ms) for the two groups, in the waking and hypnosis conditions

	Waking	Hypnosis
Low hypnotizable	522 (152)	583 (184)
High hypnotizable	579 (118)	509 (102)

RESULTS

Table 1 shows the mean stop signal reaction times for the 'highs' and 'lows' in the two conditions. Overall, 'highs' maintained shorter SSRTs, but the difference was not statistically significant. A two-way analysis of variance (state × hypnotizability) revealed only the interaction to be significant, $F_{1,12} = 11.8$, $p = 0.005$. This shows that attempting hypnosis by people who are of low susceptibility results in an increase in SSRT, whereas in those who are highly hypnotizable hypnosis lowers SSRT (i.e. increases inhibition) with respect to the waking value.

DISCUSSION

This small-scale study suggests strongly that people who are adept at hypnosis do not achieve this by engaging in a generalized reduction in inhibition. In fact the reverse appears to be true; they become better able to inhibit. This can be interpreted as being in line with the results of McGeown et al. (2009) who showed with fMRI that hypnotized 'highs' had reduced default mode activity. Of course, the relative inactivity of their participants' brains may have been attributable to lack of excitation, rather than copious inhibition, but it is implausible that the level of inhibition had been reduced.

Another parallel may be found in the results of Gruzelier, Gray and Horn (2002) who recorded evoked potentials in an odd-ball experiment. In this kind of test brain activity is monitored via scalp electrodes, while a series of identical stimuli (simple 'beeps') is presented. Each stimulus produces a neural response, the evoked potential, which is detected via electroencephalography. The unchanging stimuli produce only a small response, but when an 'odd-ball' is introduced (a beep of different pitch or duration) there is a very strong, characteristic electrical response. Gruzelier et al. found that in hypnotized 'highs' this response was dramatically reduced, whereas when 'lows' were hypnotized an enhanced response was produced.

The diminished response of the 'highs' in the Gruzelier et al. study is presumably attributable to increased inhibition, while the 'lows' appear to have reduced it. Both these effects precisely mirror those of the current experiment and appear to be part of a general tendency in 'lows' not simply to fail to produce effects, but actually to show reverse effects (Naish, 2014).

A clinical observation may be of relevance to the role of inhibition in hypnosis. It is that obsessive-compulsive patients are widely held to be very difficult to hypnotize; if formally tested they would score as 'lows'. It is also the case that these people are poor at inhibiting, so perform relatively badly on the SSRT task (Lipszyc & Schachar, 2010). If disinhibition were an aid to hypnosis, then obsessive-compulsive disorder should be associated with high susceptibility. It is not, but another condition is: post-traumatic stress disorder (PTSD). Those suffering from PTSD are more than averagely hypnotizable (Yard, et al., 2008) and appear to have good inhibition, except for trauma-related material (Naish, 2012). The flashbacks of PTSD are remarkably like hypnotic hallucinations, and for patients the focus on the trauma material combined with the

ignoring of disconfirming information can lead to the flashback being interpreted as reality, even months after the precipitating event. As with hypnotic 'highs', these effects are explicable in terms of a well-tuned deployment of inhibition and disinhibition.

If, as is being argued, strategic inhibition is an important part of achieving hypnotic effects, it is not immediately clear how consuming moderate amounts of alcohol improves hypnotic responding. The people being tested in the Semmens-Wheeler et al. (2013) study were only modestly responsive to hypnosis, but what made them so is not known. It was suggested in the introduction that hypnotic effects might be initiated via a mix of excitatory and inhibitory mechanisms. It is possible that people who are moderately susceptible are relatively good at initiating excitatory processes, sowing the seeds of a non-veridical experience, but they may be less good at turning off the disconfirming inhibition, the kind of inhibition that signals the effect to be coming from within, not without. If this were the case, then modest amounts of alcohol may facilitate the strategic inhibition reduction, although larger amounts would disinhibit too widely.

CONCLUSION

People who score high on hypnotic susceptibility scales appear to increase their ability to inhibit when they are hypnotized. This is taken to support models of hypnosis that explain the phenomenon in terms of focused attention and strategic deployment of frontal lobe involvement.

It is recognized that this is a small study, that may not generalize to lower levels of hypnotic susceptibility nor, possibly, to other means of testing for inhibition. However, it is an indication of an area deserving further exploration.

REFERENCES

Baumeister RF, Masicampo EJ (2010). Conscious thought is for facilitating social and cultural interactions: how mental simulations serve the animal-culture interface. *Psychological Review* 117: 945–71.

Blakemore S-J, Oakley DA, Frith CD (2003). Delusions of alien control in the normal brain. *Neuropsychologia* 41: 1058–1067.

Buehlmann A, Deco G (2008). The neuronal basis of attention: Rate versus synchronization modulation. *Journal of Neuroscience* 28: 7679–7686.

Doesburg SM, Roggeveen AB, Kitajo K and Ward LM (2008). Large-scale gamma-band phase synchronization and selective attention. *Cerebral Cortex* 18: 386–396.

Fingelkurts AA, Fingelkurts AA, Kallio S, Revonsuo A (2007). Cortex functional connectivity as a neurophysiological correlate of hypnosis: An EEG case study. *Neuropsychologia* 45: 1452-1462.

Gruzelier J, Gray M, Horn P (2002) The involvement of frontally modulated attention in hypnosis and hypnotic susceptibility: Cortical evoked potential evidence. *Contemporary Hypnosis* 19: 179-189.

Haig AR, Gordon E, De Pascalis V, Meares RA, Bahramali H, Harris A (2000) Gamma activity in schizophrenia: evidence of impaired network binding? *Clinical Neurophysiology* 111: 1461–1468.

Kirsch I, Cardeña E, Derbyshire S, Dienes Z, Heap M, Kallio S, Mazzoni G, Naish P, Oakley D, Potter C, Walters V and Whalley M. (2011). Definitions of hypnosis and hypnotizability and their relation to suggestion and suggesitibility: a consensus statement. *Contemporary Hypnosis and Integrative Therapy* 28: 107–11.

Kosslyn SM, Thompson WL, Costantini-Ferrando MF, Alpert NM, Spiegel D (2000). Hypnotic visual illusion alters color processing in the brain. *American Journal of Psychiatry* 157: 1279-1284.

Lawrie SM, Buechel C, Whalley HC, Frith CD, Friston KJ, Johnstone EC (2002). Reduced frontotemporal functional connectivity in schizophrenia associated with auditory hallucinations. *Biological Psychiatry* 51: 1008–1011.

Lipszyc J, Schachar R (2010). Inhibitory control and psychopathology: A meta-analysis of studies using the stop signal task. *Journal of the International Neuropsychological Society* 16: 1064–1076.

McGeown WJ, Mazzoni G, Venneri A, Kirsch I (2009). Hypnotic induction decreases anterior default mode activity. *Consciousness and Cognition* 18: 848–855.

Naish P (2012). Hypnosis: The theory behind the therapy. In L. Brann, J. Owens and A. Williamson (eds) *The Handbook of Contemporary Clinical Hypnosis*, Chichester: Wiley-Blackwell.

Naish P (2013). Hallucinations in hypnosis. In F. Macpherson and D. Platchias (eds) *Hallucination: Philosophy and Psychology*, Cambridge, MA: MIT Press.

Naish P (2014). The perception of time in hypnosis. In V. Arstila and D. Lloyd (eds) *Subjective Time: The Philosophy, Psychology, and Neuroscience of Temporality*, Cambridge, MA: MIT Press.

Semmens-Wheeler R, Dienes Z, Duka T (2013). Alcohol increases hypnotic susceptibility. *Consciousness and Cognition* 22: 1082–1091.

Shergill SS, Samson G, Bays PM, Frith CD, Wolpert DM (2005). Evidence for sensory prediction deficits in schizophrenia. *American Journal of Psychiatry* 162: 2384–2386

Shor RE, Orne EC (1962). *Harvard Group Scale of Hypnotic Susceptibility, Form A,* Palo Alto, CA: Consulting Psychologists Press.

Spanos N (1986). Hypnotic behaviour: A social–psychological interpretation of amnesia, analgesia, and 'trance logic'. *Behavioural and Brain Sciences* 9: 449–502.

Szechtman H, Woody E, Bowers KS, Nahmias C.(1998). Where the imaginal appears real: A positron emission tomography study of auditory hallucinations. *Proceedings of the National Academy of Sciences* 95: 1956–1960.

van Gaal S, Lamme VAF (2012). Unconscious high-level information processing: Implication for neurobiological theories of consciousness. *The Neuroscientist* 18: 287–301.

Yard SS, DuHamel KN, Galynker II (2008). Hypnotizability as a potential risk factor for post-traumatic stress: A review of quantitative studies. *International Journal of Clinical and Experimental Hypnosis* 56: 334–356.

Correspondence to: Peter L. N. Naish, Department of Psychology, The Open University, Milton Keynes MK7 6AA, UK
Email: peter.naish@open.ac.uk
Phone: +44 (0)1491 680 944